SnapShot: Root Development

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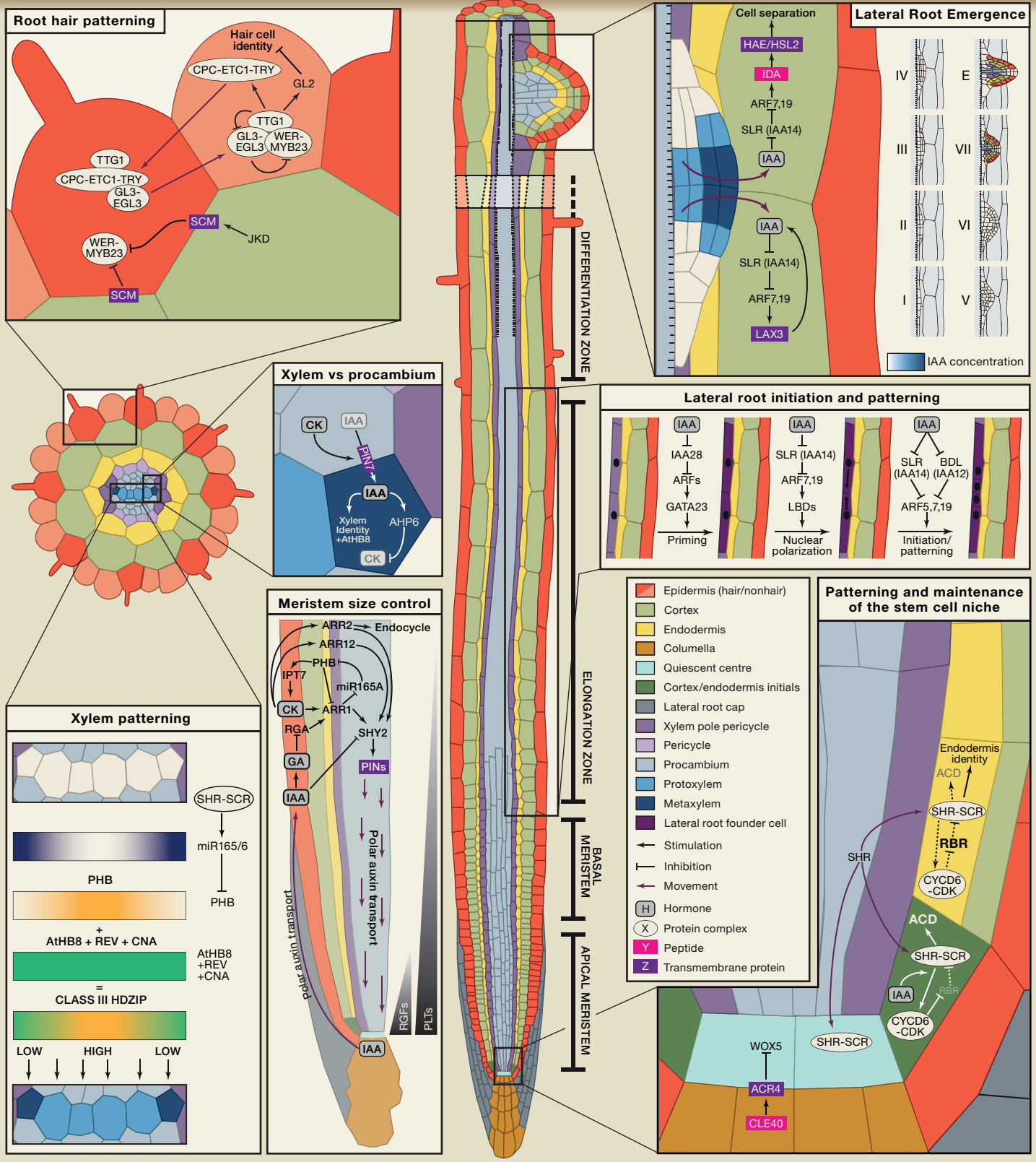
Cell



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Cell

Patterning and Maintenance of the Stem Cell Niche

Plant roots are sustained by the continuous cell division and development of the root meristem. An organizing center termed the quiescent center (QC), located at the tip of the apical meristem, maintains the identity of surrounding stem cells by expression of *WOX5*. Differentiated columella cells express the peptide *CLE40*, which promotes differentiation via the receptor-like kinase ACR4 to regulate the activity of WOX5, which can act redundantly with the SHR/SCR and PLT pathways to maintain the stem cell niche. *SHR* mRNA is expressed in the stele, but the protein translocates to surrounding cells in the QC and endodermis, activating SCR expression. SCR interacts with SHR to regulate its nuclear localization. In addition to QC maintenance, the SHR:SCR complex is required for endodermal and cortical differentiation, tissues that originate from the same stem cell, the cortex/endodermis initial (CEI). In the presence of auxin, the SHR:SCR complex directly activates *CYCD6;1* expression and promotes asymmetric cell division in the CEI or its daughter cell. The CYCD6;1-CDK complex inactivates the retinoblastoma-related RBR protein through phosphorylation, and RBR can, in turn, directly inhibit the complex. This network creates a position-dependent fine-tuning mechanism for stem cell maintenance and differentiation.

Meristem Size Control

After several rounds of division, cells transition toward expansion in the basal meristem, exit the cell cycle, and acquire tissue-specific characteristics in the elongation and differentiation zones. Polar auxin transport by AUX/LAX, ABCB, and PIN classes of auxin influx and efflux carriers creates an auxin maximum at the root apical meristem controlling meristem activity and size, together with gradients of PLT transcription factors and RGF sulfated peptides. Hormonal crosstalk between auxin (IAA), cytokinin (CK), and gibberellin (GA) regulates the expression of *SHY2/IAA3*, a member of the Aux/IAA family of auxin response repressors, as cells transition between meristem and elongation zones. Enhancement of *SHY2/IAA3* inhibits auxin response and PIN expression, causing cell differentiation. CK also promotes a switch from mitosis to the endocycle (i.e., cell enlargement and DNA replication without division). The transcription factor *PHB* activates the biosynthesis gene *IPT7*; CK signaling represses both *PHB* and microRNA165 (an inhibitor of *PHB* mRNA), creating a feedback loop and providing robustness against fluctuations in CK levels. These signaling outputs promote increased root cell volume and differentiation.

Xylem Patterning

The vascular tissues provide a long-distance transport mechanism for water and nutrients. In *Arabidopsis*, the vascular cylinder has a single xylem axis with protoxylem cells at the marginal positions and metaxylem cells in the central positions. This axis is flanked by procambial stem cells and two phloem poles. A domain of high auxin response defines the xylem axis and promotes the expression of xylem identity genes such as *AtHB8*. In contrast, the procambial/phloem cells are represented by a domain of high CK signaling output. A sharp boundary between these two signaling domains is maintained by a mutually inhibitory mechanism whereby auxin promotes the expression of the CK-signaling inhibitor *AHP6* and high CK response promotes the activity of a group of auxin efflux transporters (including PIN7) that direct auxin out of the procambial cells. The specification of protoxylem versus metaxylem identity is determined by the combined gene dosage of five class III HD-ZIP transcription factors—*CNA*, *REV, AtHB8*, *PHV*, and, in particular, *PHB*—with cells with the highest level of HD-ZIP expression becoming metaxylem. The formation of the SHR:SCR complex in the endodermis drives the expression of mobile microRNA165/6, which diffuses into the vascular cylinder and degrades *PHB* mRNA to create a gradient of *PHB* with its maxima in the central cells.

Root Hair Patterning

Root hairs are key for water and nutrient uptake and for soil anchoring. The default fate for root epidermis is to become a root hair cell, and positional cues from the underlying cortical cells (green) are important in its acquisition. Epidermal cells communicate to promote nonhair cell fate in one cell (dark red) and repress this fate in its neighbors (light red). Hair cell fate is repressed by a complex of the transcription factors *WER/MYB23*, *GL3/EGL3*, and *TTG1*, which directly activates transcription of the hair cell fate repressor GL2. This complex directly activates transcription of its inhibitor complex *CPC/TRY/ETC1*, which moves to neighboring cells to replace WER/MYB23 to inactivate the activator complex. Cortical cells provide additional levels of root hair cell fate regulation. The transcription factor JKD activates the epidermal leucine-rich repeat (LRR) receptor-kinase-like protein SCM via an unknown mechanism that can indirectly repress the transcription of WER in root hair epidermal cells. The cortical transcription factor *SCZ* can also repress root hair cell fate in cortical cells and can induce the separation of epidermal cell fate by a non-cell-autonomous mechanism.

Lateral Root Initiation and Patterning

Root branching is critical to adapting to local soil environments. *Arabidopsis* lateral roots (LRs) originate from a subset of pericycle cells adjacent to the xylem pole that are "primed" in the basal meristem by rhythmic activation of the auxin response in this zone, which stimulates nuclear migration toward the common cell wall in pairs of pericycle cells. The resulting asymmetric cell division creates two central daughter cells and larger flanking cells with different cell fates. The daughter cells continue to divide and create a dome-shaped LR primordium that eventually forms a new meristem. Several Aux/IAA-ARF modules regulate activation of target genes, including transcription factors such as GATA23 for priming and auxin-inducible LBD/ASLs for nuclear polarization.

Lateral Root Emergence

As LRs originate from pericycle cells, new primordia have to emerge through overlaying tissues by triggering cell separation. This is induced by auxin release from new primordia and local activation of the auxin response in overlying endodermal, cortical, and epidermal cells. In cortical cells, auxin induces the auxin influx carrier LAX3 to create a positive-feedback loop that reinforces auxin influx specifically into cells overlying the emerging primordia. Auxin induces expression of a secreted peptide, IDA, which binds the LRR receptor-like kinases HAE and HSL2 and upregulates several cell-wall-remodeling enzymes that promote cell separation.

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